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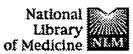
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A new aspartyl protease on 21q22.3, BACE2, is highly similar to Alzheimer's amyloid precursor protein beta-secretase.

Solans A, Estivill X, de La Luna S.

Down Syndrome Research Group, Medical and Molecular Genetics Center, IRO, Hospital Duran i Reynals, Barcelona, Spain.

Down syndrome individuals develop abnormalities of most organs, including all the pathological and neurochemical features of Alzheimer's disease, by the early age of 30 yr. Here, we report the isolation and characterization of BACE2, a gene mapping on human chromosome 21q22.3, which is highly similar to a transmembrane aspartyl protease, BACE (for beta-site APPcleaving enzyme), which is able to catalyze the beta-secretase cleavage of Alzheimer's amyloid precursor protein (APP). BACE2 is expressed in a wide variety of organs and tissues, with several transcripts due to alternative splicing and the use of two polyadenylation signals. The BACE2 gene product is a 518 amino acid protein with the signature of an aspartic protease, a 20residue signal peptide, and two putative N-glycosylation sites. In addition, and similarly to BACE, BACE2 differs from the other members of the human aspartic protease family in the number and distribution of putative disulfide bonds and in the presence of an extended C-terminal region which contains a predicted transmembrane segment. BACE2 could be involved in the Alzheimer-like neuropathology of Down syndrome, as well as in Alzheimer's disease linked to chromosome 21 but not showing mutations in APP. Copyright 2000 S. Karger AG, Basel.

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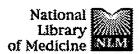
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Regulation of APP cleavage by alpha-, beta- and gammasecretases.

Nunan J, Small DH.

Laboratory of Molecular Neurobiology, Department of Pathology, University of Melbourne, 3010, Melbourne, Vic., Australia.

Proteolytic cleavage of the amyloid protein from the amyloid protein precursor (APP) by APP secretases is a key event in Alzheimer's disease (AD) pathogenesis. alpha-Secretases cleave APP within the amyloid sequences, whereas beta- and gamma-secretases cleave on the N- and C-terminal ends respectively. The transmembrane aspartyl protease BACE has been identified as beta-secretase and several proteases (ADAM-10, TACE, PC7) may be alpha-secretases. A number of studies have suggested that presenilins could be gamma-secretases, although this remains to be demonstrated conclusively. Inhibition of beta- and gamma-secretase, or stimulation of alpha-secretase, is a rational strategy for therapeutic intervention in AD.

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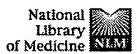
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Specificity of memapsin 1 and its implications on the design of memapsin 2 (beta-secretase) inhibitor selectivity.

Turner RT 3rd, Loy JA, Nguyen C, Devasamudram T, Ghosh AK, Koelsch G, Tang J.

Protein Studies Program, Oklahoma Medical Research Foundation and Department of Biochemistry and Molecular Biology, University of Oklahoma Health Sciences Center, Oklahoma City, Oklahoma 73104, USA.

Memapsin 1 is closely homologous to memapsin 2 (BACE), or beta-secretase. whose action on beta-amyloid precursor protein (APP) leads to the production of beta-amyloid (A beta) peptide and the progression of Alzheimer's disease. Memapsin 2 is a current target for the development of inhibitor drugs to treat Alzheimer's disease. Although memapsin 1 hydrolyzes the beta-secretase site of APP, it is not significantly present in the brain, and no direct evidence links it to Alzheimer's disease. We report here the residue specificity of eight memapsin 1 subsites. In substrate positions P(4), P(3), P(2), P(1), P(1)', P(2)', P(3)', and P(4)', the most preferred residues are Glu, Leu, Asn, Phe, Met, Ile, Phe, and Trp, respectively, while the second preferred residues are Gln, Ile, Asp, Leu, Leu, Val, Trp, and Phe, respectively. Other less preferred residues can also be accommodated in these subsites of memapsin 1. Despite the broad specificity, these residue preferences are strikingly similar to those of human memapsin 2 [Turner et al. (2001) Biochemistry 40, 10001-10006] and thus pose a serious problem to the design of differentially selective inhibitors capable of inhibiting memapsin 2. This difficulty was confirmed by the finding that several potent memapsin 2 inhibitors effectively inhibited memapsin 1 as well. Several possible approaches to overcome this problem are discussed.

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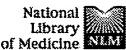
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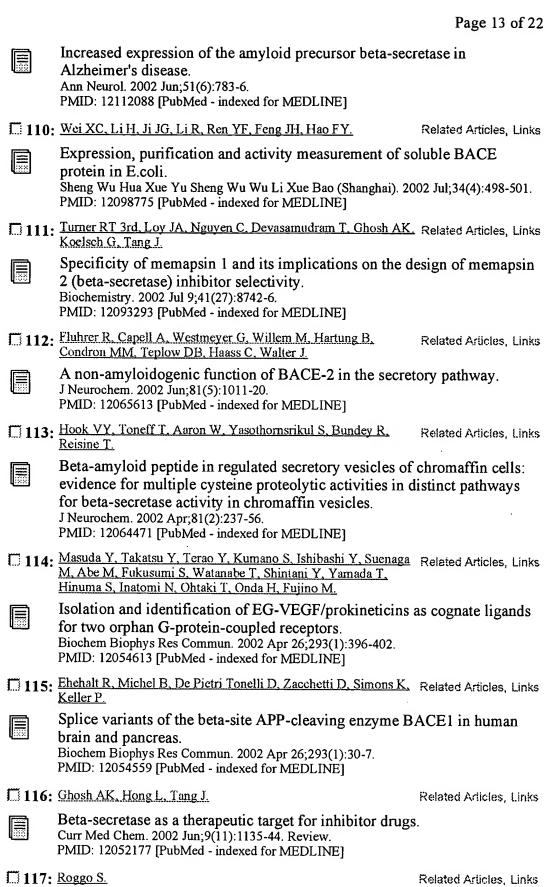
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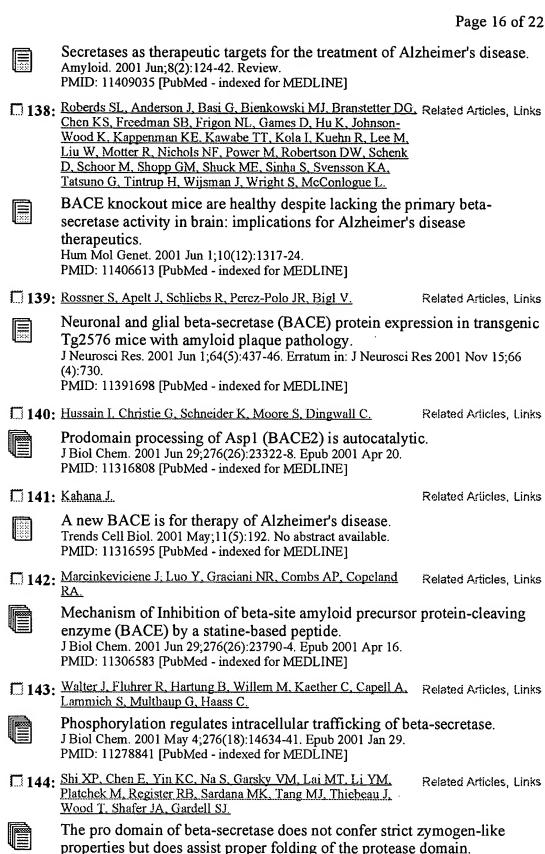
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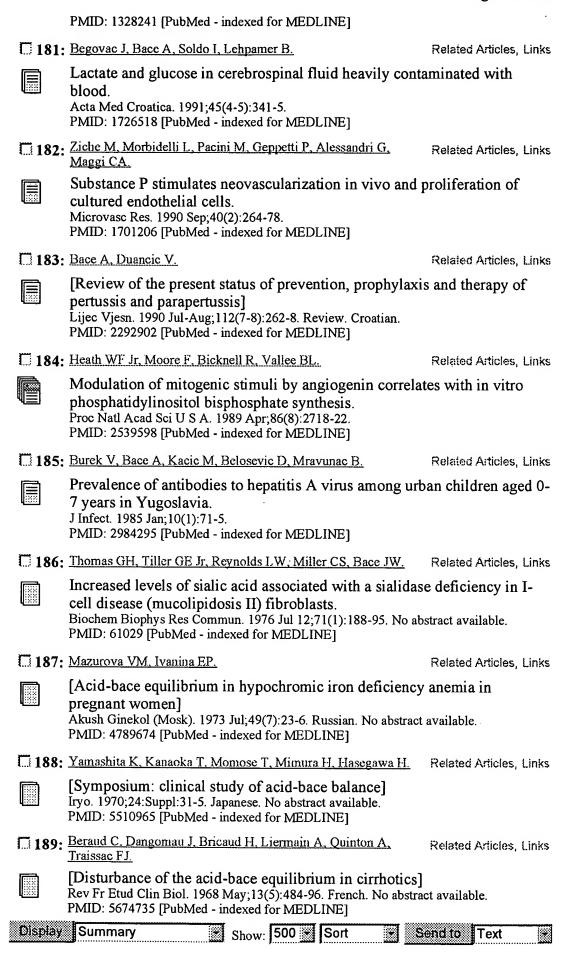
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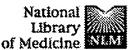
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Department of Pathology, Johns Hopkins University School of Medicine, Baltimore, MD 21205-2196.

We assessed the ability of a murine monoclonal antibody to bind selectively to beta-amyloid in the brains of living nonhuman primates. To circumvent the blood-brain barrier, we injected unlabeled antibody 10D5 (murine whole IgG1 and/or Fab fragments) into the cerebrospinal fluid of the cisterna magna in three aged monkeys. A control animal was given an intracisternal injection of nonimmune mouse whole IgG plus Fab. Twenty-four hours later, the animals were perfused and prepared for immunohistochemical detection of bound murine immunoglobulin in brain. All three experimental animals showed selective binding of 10D5 to approximately 5-15% of amyloid deposits in cerebral cortex, primarily near the cortical surface. There was no labeling in the control animal. In vivo-labeled deposits were confirmed to be betaamyloid by electron microscopy and by in vitro immunohistochemistry in adjacent sections. The animals tolerated the injection well, although some polymorphonuclear leukocytes infiltrated portions of the subarachnoid space and superficial neocortex. These results provide the first demonstration that it may be feasible to selectively direct a tagged monoclonal antibody to betaamyloid in the brain for therapeutic or diagnostic purposes. With enhancement of labeling efficiency, the method also may be useful for studying the progression of beta-amyloidosis in experimental animals using emission tomography.

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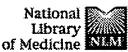
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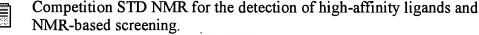
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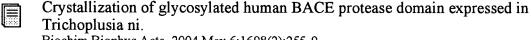
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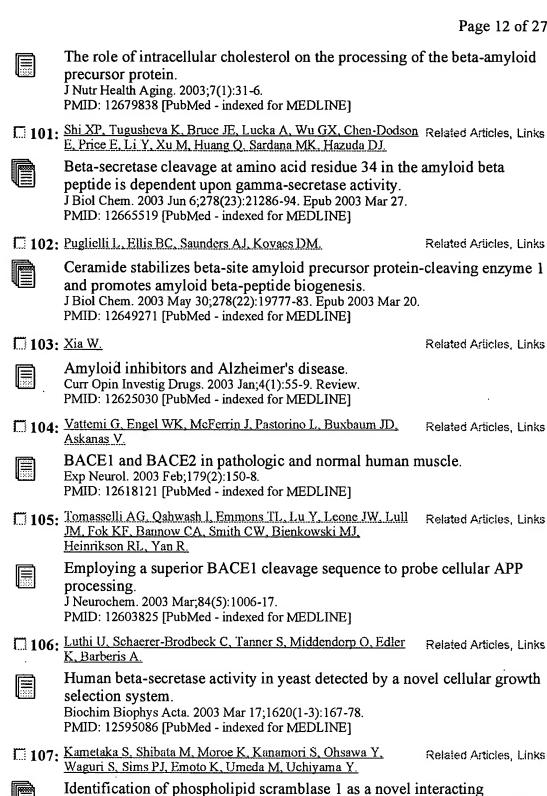
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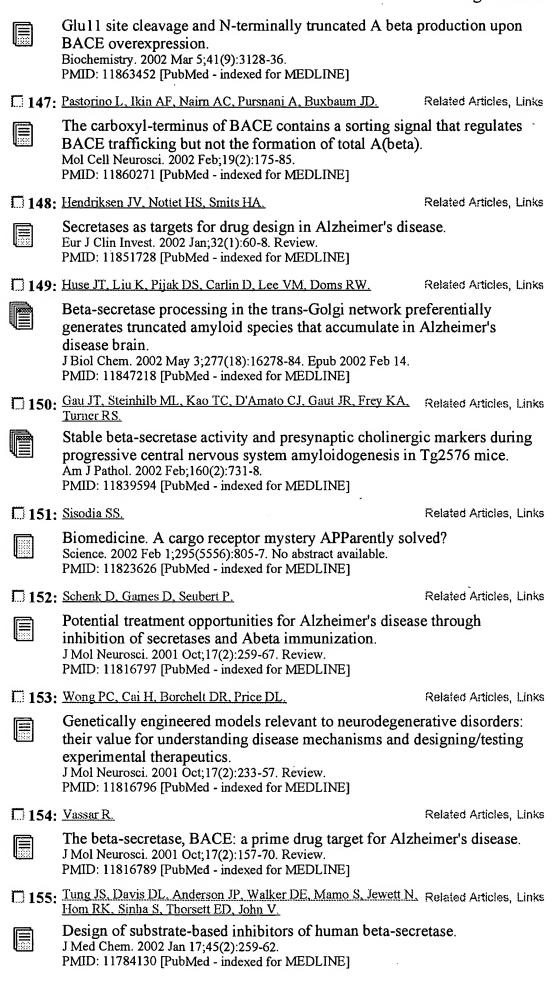
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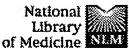
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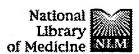
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Levels of alpha- and beta-secretase cleaved amyloid precursor protein in the cerebrospinal fluid of Alzheimer's disease patients.

Sennvik K, Fastbom J, Blomberg M, Wahlund LO, Winblad B, Benedikz

Division of Geriatric Medicine, Karolinska Institute, NEUROTEC, KFC NOVUM, Huddinge, Sweden.

Alternative cleavage of the amyloid precursor protein (APP) results in generation and secretion of both soluble APP (sAPP) and beta-amyloid (Abeta). Abeta is the main component of the amyloid depositions in the brains of Alzheimer's disease (AD) patients. Using Western blotting, we compared the levels of alpha-secretase cleaved sAPP, beta-secretase cleaved sAPP and total sAPP, in cerebrospinal fluid (CSF) from 13 sporadic AD patients and 13 healthy controls. Our findings show significant amounts of beta-secretase cleaved sAPP in CSF. There was no statistically significant difference in the levels of beta-secretase cleaved sAPP between AD patients and controls. The levels of alpha-secretase cleaved sAPP and total sAPP were, however, found to be significantly lower in the AD patients than in the controls.

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Human aspartic protease memapsin 2 cleaves the beta-secretase site of beta-amyloid precursor protein.

Lin X, Koelsch G, Wu S, Downs D, Dashti A, Tang J.

Protein Studies Program, Oklahoma Medical Research Foundation, and Department of Biochemistry, University of Oklahoma Health Sciences Center, Oklahoma City, OK 73104, USA.

The cDNAs of two new human membrane-associated aspartic proteases, memapsin 1 and memapsin 2, have been cloned and sequenced. The deduced amino acid sequences show that each contains the typical pre, pro, and aspartic protease regions, but each also has a C-terminal extension of over 80 residues, which includes a single transmembrane domain and a C-terminal cytosolic domain. Memapsin 2 mRNA is abundant in human brain. The protease domain of memapsin 2 cDNA was expressed in Escherichia coli and was purified. Recombinant memapsin 2 specifically hydrolyzed peptides derived from the beta-secretase site of both the wild-type and Swedish mutant beta-amyloid precursor protein (APP) with over 60-fold increase of catalytic efficiency for the latter. Expression of APP and memapsin 2 in HeLa cells showed that memapsin 2 cleaved the beta-secretase site of APP intracellularly. These and other results suggest that memapsin 2 fits all of the criteria of betasecretase, which catalyzes the rate-limiting step of the in vivo production of the beta-amyloid (Abeta) peptide leading to the progression of Alzheimer's disease. Recombinant memapsin 2 also cleaved a peptide derived from the processing site of presenilin 1, albeit with poor kinetic efficiency. Alignment of cleavage site sequences of peptides indicates that the specificity of memapsin 2 resides mainly at the S(1)' subsite, which prefers small side chains such as Ala, Ser, and Asp.

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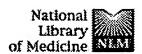
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Cellular and molecular basis of beta-amyloid precursor protein metabolism.

Greenfield JP, Gross RS, Gouras GK, Xu H.

Fisher Center for Alzheimer Research and Laboratory of Molecular and Cellular Neuroscience, The Rockefeller University, New York NY 10021, USA.

In molecular neurobiology, perhaps no molecule has been as thoroughly examined as Alzheimer's beta-amyloid precursor protein (beta-APP). In the years since the cDNA encoding beta-APP was cloned, the protein has been the subject of unparalleled scrutiny on all levels. From molecular genetics and cellular biology to neuroanatomy and epidemiology, no scientific discipline has been left unexplored - and with good reason, beta-amyloid (Abeta) is the main constituent of the amyloidogenic plaques which are a primary pathological hallmark of Alzheimer's disease, and bta-APP is the protein from which Abeta is cleaved and released. Unraveling the molecular events underlying Abeta generation has been, and remains, of paramount importance to scientists in our field. In this review we will trace the progress that has been made in understanding the molecular and cellular basis of beta-APP trafficking and processing, or alternatively stated, the molecular basis for Abeta generation. Imperative to a complete understanding of Abeta generation is the delineation of its subcellular localization and the identification of proteins that play either direct or accessory roles in Abeta generation. We will focus on the regulation of beta-APP cleavage through diverse signal transduction mechanisms and discuss possible points of therapeutic intercession in what has been postulated to be a seminal molecular step in the cascade of events terminating in the onset of dementia, loss of neurons, and eventual death from Alzheimer's disease.

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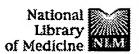
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Cellular and molecular basis of beta-amyloid precursor protein metabolism.

Greenfield JP, Gross RS, Gouras GK, Xu H.

Fisher Center for Alzheimer Research and Laboratory of Molecular and Cellular Neuroscience, The Rockefeller University, New York NY 10021, USA.

In molecular neurobiology, perhaps no molecule has been as thoroughly examined as Alzheimer's beta-amyloid precursor protein (beta-APP). In the years since the cDNA encoding beta-APP was cloned, the protein has been the subject of unparalleled scrutiny on all levels. From molecular genetics and cellular biology to neuroanatomy and epidemiology, no scientific discipline has been left unexplored - and with good reason, beta-amyloid (Abeta) is the main constituent of the amyloidogenic plaques which are a primary pathological hallmark of Alzheimer's disease, and bta-APP is the protein from which Abeta is cleaved and released. Unraveling the molecular events underlying Abeta generation has been, and remains, of paramount importance to scientists in our field. In this review we will trace the progress that has been made in understanding the molecular and cellular basis of beta-APP trafficking and processing, or alternatively stated, the molecular basis for Abeta generation. Imperative to a complete understanding of Abeta generation is the delineation of its subcellular localization and the identification of proteins that play either direct or accessory roles in Abeta generation. We will focus on the regulation of beta-APP cleavage through diverse signal transduction mechanisms and discuss possible points of therapeutic intercession in what has been postulated to be a seminal molecular step in the cascade of events terminating in the onset of dementia, loss of neurons, and eventual death from Alzheimer's disease.

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Modeling of substrate specificity of the Alzheimer's disease amyloid precursor protein beta-secretase.

Sauder JM, Arthur JW, Dunbrack RL Jr.

Fox Chase Cancer Center, Institute for Cancer Research, 7701 Burholme Avenue, Philadelphia, PA 19111, USA.

The enzyme BACE (beta-site APP-cleaving enzyme) has recently been identified as the beta-secretase that cleaves the amyloid precursor protein (APP) to produce the N terminus of the Abeta peptide found in plagues in the brains of Alzheimer's disease patients. BACE is an aspartic protease similar to pepsin and renin. Comparative modeling of the three-dimensional structure of BACE in complex with its substrate shows that several residues confer specificity of the enzyme for APP. In particular, Arg296 forms a salt-bridge with the P1' Asp of the APP substrate, explaining the unusual preference of BACE among aspartic proteases for a P1' residue that is negatively charged. Several hydrophobic residues in the enzyme form a pocket for the P1 hydrophobic residue (Met in wild-type APP and Leu in APP with the "Swedish mutation" associated with early-onset of Alzheimer's disease). Inhibitors that can bind to the BACE active site may prove useful for drugs to treat and prevent Alzheimer's disease. Copyright 2000 Academic Press.

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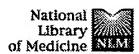
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Maturation and endosomal targeting of beta-site amyloid precursor protein-cleaving enzyme. The Alzheimer's disease beta-secretase.

Huse JT, Pijak DS, Leslie GJ, Lee VM, Doms RW.

Department of Pathology and Laboratory Medicine, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania 19104, USA.

The amyloidogenic Abeta peptide is liberated from the amyloid precursor protein (APP) by two proteolytic activities, beta-secretase and gammasecretase. Recently, a type I membrane protein termed BACE (beta-site APP cleaving enzyme) with characteristics of an aspartyl protease has been identified as the beta-secretase. We undertook a series of biochemical and morphological investigations designed to characterize the basic properties of this protein. Initial studies indicated that BACE undergoes N-linked glycosylation at three of four potential sites. Metabolic pulse-chase experiments revealed that after core glycosylation, BACE is rapidly and efficiently transported to the Golgi apparatus and distal secretory pathway. BACE was also found to be quite stable, being turned over with a t(12) of approximately 16 h. Retention of BACE in the endoplasmic reticulum by introduction of a C-terminal dilysine motif prevented complex carbohydrate processing and demonstrated that propertide cleavage occurs after exit from this organelle. BACE exhibited intramolecular disulfide bonding but did not form oligomeric structures by standard SDS-polyacrylamide gel electrophoresis analysis and sedimented as a monomer in sucrose velocity gradients. Immunofluorescence studies showed a largely vesicular staining pattern for BACE that colocalized well with endosomal, but not lysosomal, markers. Measurable levels of BACE were also detected on the plasma membrane by both immunostaining and cell surface biotinylation, and cycling of the protein between the cell membrane and the endosomes was documented. A cytoplasmic dileucine motif was found to be necessary for normal targeting of BACE to the endosomal system and accumulation of the protein in this intracellular site.

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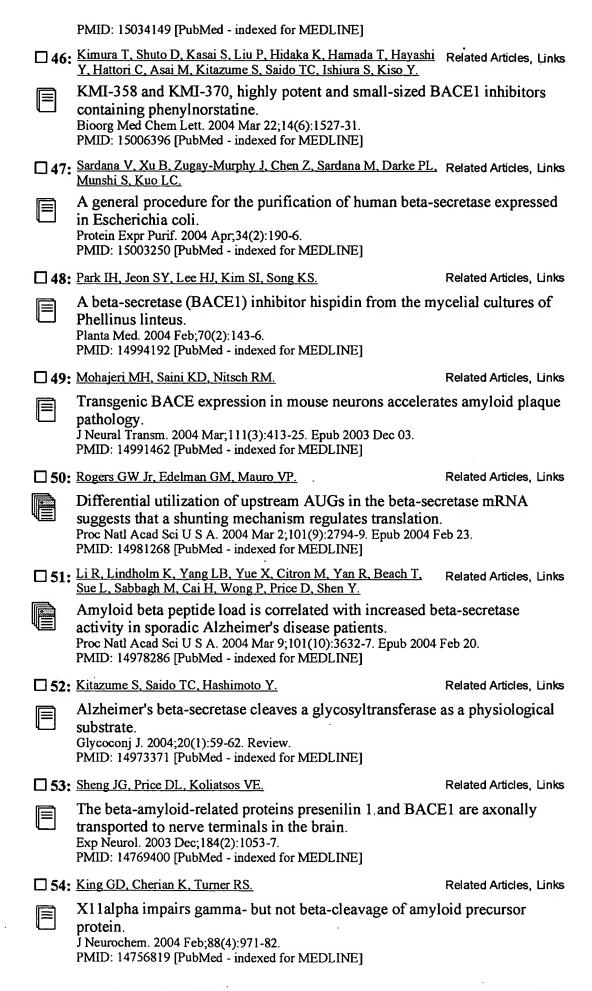
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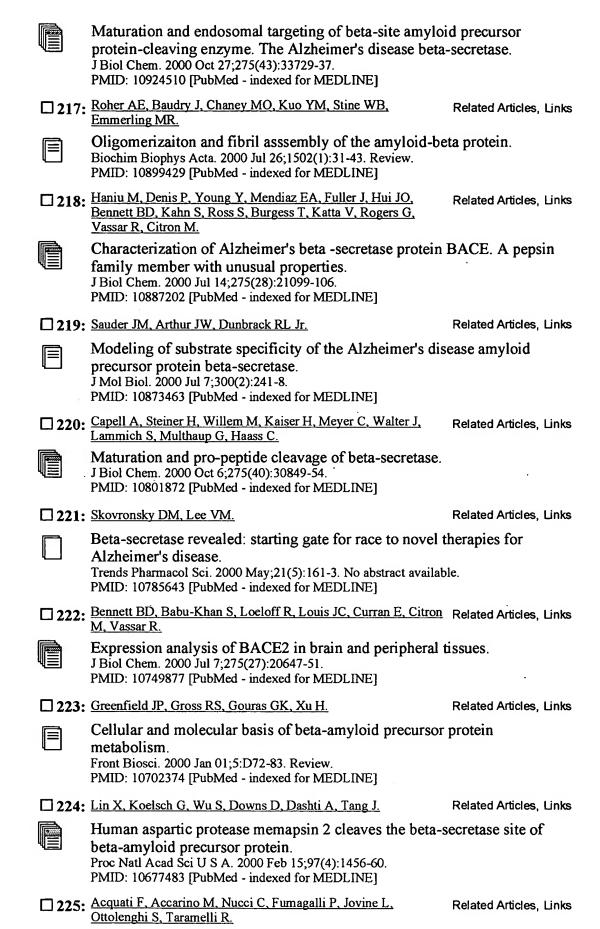
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- Science. 1999 Nov 12;286(5443):1254. Science. 1999 Oct 22;286(5440):650-1.
- Full text article at ww.sciencomag.org

Beta-secretase cleavage of Alzheimer's amyloid precursor protein by the transmembrane aspartic protease BACE.

Vassar R, Bennett BD, Babu-Khan S, Kahn S, Mendiaz EA, Denis P, Teplow DB, Ross S, Amarante P, Loeloff R, Luo Y, Fisher S, Fuller J, Edenson S, Lile J, Jarosinski MA, Biere AL, Curran E, Burgess T, Louis JC, Collins F, Treanor J, Rogers G, Citron M.

Amgen, Inc., One Amgen Center Drive, M/S 29-2-B, Thousand Oaks, CA 91320-1799, USA.

Cerebral deposition of amyloid beta peptide (Abeta) is an early and critical feature of Alzheimer's disease. Abeta generation depends on proteolytic cleavage of the amyloid precursor protein (APP) by two unknown proteases: beta-secretase and gamma-secretase. These proteases are prime therapeutic targets. A transmembrane aspartic protease with all the known characteristics of beta-secretase was cloned and characterized. Overexpression of this protease, termed BACE (for beta-site APP-cleaving enzyme) increased the amount of beta-secretase cleavage products, and these were cleaved exactly and only at known beta-secretase positions. Antisense inhibition of endogenous BACE messenger RNA decreased the amount of beta-secretase cleavage products, and purified BACE protein cleaved APP-derived substrates with the same sequence specificity as beta-secretase. Finally, the expression pattern and subcellular localization of BACE were consistent with that expected for beta-secretase. Future development of BACE inhibitors may prove beneficial for the treatment of Alzheimer's disease.

PMID: 10531052 [PubMed - indexed for MEDLINE]

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Identification of a novel aspartic protease (Asp 2) as betasecretase.

Hussain I, Powell D, Howlett DR, Tew DG, Meek TD, Chapman C, Gloger IS, Murphy KE, Southan CD, Ryan DM, Smith TS, Simmons DL, Walsh FS, Dingwall C, Christie G.

Department of Neurosciences, SmithKline Beecham Pharmaceuticals, Harlow. Essex, United Kingdom.

The Alzheimer's disease beta-amyloid peptide (Abeta) is produced by excision from the type 1 integral membrane glycoprotein amyloid precursor protein (APP) by the sequential actions of beta- and then gamma-secretases. Here we report that Asp 2, a novel transmembrane aspartic protease, has the key activities expected of beta-secretase. Transient expression of Asp 2 in cells expressing APP causes an increase in the secretion of the N-terminal fragment of APP and an increase in the cell-associated C-terminal beta-secretase APP fragment. Mutation of either of the putative catalytic aspartyl residues in Asp 2 abrogates the production of the fragments characteristic of cleavage at the beta-secretase site. The enzyme is present in normal and Alzheimer's disease (AD) brain and is also found in cell lines known to produce Abeta. Asp 2 localizes to the Golgi/endoplasmic reticulum in transfected cells and shows clear colocalization with APP in cells stably expressing the 751-amino-acid isoform of APP.

PMID: 10656250 [PubMed - indexed for MEDLINE]

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• Nature. 1999 Dec 2;402(6761):471-2.

nature

☐ 1: Nature. 1999 Dec 2;402(6761):533-7.

Membrane-anchored aspartyl protease with Alzheimer's disease beta-secretase activity.

Yan R, Bienkowski MJ, Shuck ME, Miao H, Tory MC, Pauley AM, Brashier JR, Stratman NC, Mathews WR, Buhl AE, Carter DB, Tomasselli AG, Parodi LA, Heinrikson RL, Gurney ME.

Cell & Molecular Biology, Pharmacia & Upjohn, Inc., Kalamazoo, MI 49007, USA. rigiang.yan@am.pnu.com

Mutations in the gene encoding the amyloid protein precursor (APP) cause autosomal dominant Alzheimer's disease. Cleavage of APP by unidentified proteases, referred to as beta- and gamma-secretases, generates the amyloid beta-peptide, the main component of the amyloid plaques found in Alzheimer's disease patients. The disease-causing mutations flank the protease cleavage sites in APP and facilitate its cleavage. Here we identify a new membrane-bound aspartyl protease (Asp2) with beta-secretase activity. The Asp2 gene is expressed widely in brain and other tissues. Decreasing the expression of Asp2 in cells reduces amyloid beta-peptide production and blocks the accumulation of the carboxy-terminal APP fragment that is created by beta-secretase cleavage. Solubilized Asp2 protein cleaves a synthetic APP peptide substrate at the beta-secretase site, and the rate of cleavage is increased tenfold by a mutation associated with early-onset Alzheimer's disease in Sweden. Thus, Asp2 is a new protein target for drugs that are designed to block the production of amyloid beta-peptide peptide and the consequent formation of amyloid plaque in Alzheimer's disease.

PMID: 10591213 [PubMed - indexed for MEDLINE]

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• Nature. 1999 Dec 2:402(6761):471-2.

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Purification and cloning of amyloid precursor protein betasecretase from human brain.

Sinha S, Anderson JP, Barbour R, Basi GS, Caccavello R, Davis D, Doan M, Dovey HF, Frigon N, Hong J, Jacobson-Croak K, Jewett N, Keim P, Knops J, Lieberburg I, Power M, Tan H, Tatsuno G, Tung J, Schenk D, Seubert P, Suomensaari SM, Wang S, Walker D, John V, et al.

Elan Pharmaceuticals, South San Francisco, California 94080, USA. ssinha@elanpharama.com

Proteolytic processing of the amyloid precursor protein (APP) generates amyloid beta (Abeta) peptide, which is thought to be causal for the pathology and subsequent cognitive decline in Alzheimer's disease. Cleavage by betasecretase at the amino terminus of the Abeta peptide sequence, between residues 671 and 672 of APP, leads to the generation and extracellular release of beta-cleaved soluble APP, and a corresponding cell-associated carboxyterminal fragment. Cleavage of the C-terminal fragment by gamma-secretase (s) leads to the formation of Abeta. The pathogenic mutation K670M671->N670L671 at the beta-secretase cleavage site in APP, which was discovered in a Swedish family with familial Alzheimer's disease, leads to increased betasecretase cleavage of the mutant substrate. Here we describe a membranebound enzyme activity that cleaves full-length APP at the beta-secretase cleavage site, and find it to be the predominant beta-cleavage activity in human brain. We have purified this enzyme activity to homogeneity from human brain using a new substrate analogue inhibitor of the enzyme activity, and show that the purified enzyme has all the properties predicted for betasecretase. Cloning and expression of the enzyme reveals that human brain beta-secretase is a new membrane-bound aspartic proteinase.

PMID: 10591214 [PubMed - indexed for MEDLINE]

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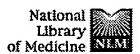
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1: J Biol Chem. 2000 Jul 14;275(28):21099-106.

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Characterization of Alzheimer's beta -secretase protein BACE. A pepsin family member with unusual properties.

Haniu M, Denis P, Young Y, Mendiaz EA, Fuller J, Hui JO, Bennett BD, Kahn S, Ross S, Burgess T, Katta V, Rogers G, Vassar R, Citron M.

Amgen Inc., Thousand Oaks, California 91320-1799, USA. mhaniu@amgen.com

The cerebral deposition of amyloid beta-peptide is an early and critical feature of Alzheimer's disease. Amyloid beta-peptide is released from the amyloid precursor protein by the sequential action of two proteases, beta-secretase and gamma-secretase, and these proteases are prime targets for therapeutic intervention. We have recently cloned a novel aspartic protease, BACE, with all the known properties of beta-secretase. Here we demonstrate that BACE is an N-glycosylated integral membrane protein that undergoes constitutive Nterminal processing in the Golgi apparatus. We have used a secreted Fc fusion-form of BACE (BACE-IgG) that contains the entire ectodomain for a detailed analysis of posttranslational modifications. This molecule starts at Glu(46) and contains four N-glycosylation sites (Asn(153), Asn(172), Asn (223), and Asn(354)). The six Cys residues in the ectodomain form three intramolecular disulfide linkages (Cys(216)-Cys(420), Cys(278)-Cys(443), and Cys(330)-Cys(380)). Despite the conservation of the active site residues and the 30-37% amino acid homology with known aspartic proteases, the disulfide motif is fundamentally different from that of other aspartic proteases. This difference may affect the substrate specificity of the enzyme. Taken together, both the presence of a transmembrane domain and the unusual disulfide bond structure lead us to conclude that BACE is an atypical pepsin family member.

PMID: 10887202 [PubMed - indexed for MEDLINE]

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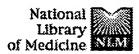
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Expression analysis of BACE2 in brain and peripheral tissues.

Bennett BD, Babu-Khan S, Loeloff R, Louis JC, Curran E, Citron M, Vassar R.

Amgen, Inc., Thousand Oaks, California 91320-1799, USA.

Beta-site amyloid precursor protein cleaving enzyme (BACE) is a novel transmembrane aspartic protease that possesses all the known characteristics of the beta-secretase involved in Alzheimer's disease (Vassar, R., Bennett, B. D., Babu-Khan, S., Kahn, S., Mendiaz, E. A., Denis, P., Teplow, D. B., Ross, S., Amarante, P., Loeloff, R., Luo, Y., Fisher, S., Fuller, J., Edenson, S., Lile, J., Jarosinski, M. A., Biere, A. L., Curran, E., Burgess, T., Louis, J. -C., Collins, F., Treanor, J., Rogers, G., and Citron, M. (1999) Science 286, 735-.741). We have analyzed the sequence and expression pattern of a BACE homolog termed BACE2. BACE and BACE2 are unique among aspartic proteases in that they possess a carboxyl-terminal extension with a predicted transmembrane region and together they define a new family. Northern analysis reveals that BACE2 mRNA is expressed at low levels in most human peripheral tissues and at higher levels in colon, kidney, pancreas, placenta, prostate, stomach, and trachea. Human adult and fetal whole brain and most adult brain subregions express very low or undetectable levels of BACE2 mRNA. In addition, in situ hybridization of adult rat brain shows that BACE2 mRNA is expressed at very low levels in most brain regions. The very low or undetectable levels of BACE2 mRNA in the brain are not consistent with the expression pattern predicted for beta-secretase.

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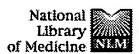
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Inhibitors of beta-amyloid formation based on the beta-secretase cleavage site.

Abbenante G, Kovacs DM, Leung DL, Craik DJ, Tanzi RE, Fairlie DP.

Centre for Drug Design and Development, University of Queensland, Brisbane, Queensland, 4072, Australia.

A series of inhibitors of beta-amyloid formation have been developed based on the beta-secretase cleavage site (VNL-DA) of the Swedish mutant Amyloid Precursor Protein. A simple tripeptide aldehyde was found to be the most potent (IC(50) = 700 nM) in the series displaying an inhibitory profile which is different from reported inhibitors of beta-amyloid formation. Copyright 2000 Academic Press.

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1: Nat Biotechnol. 2000 Jan; 18(1):66-70.

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Identification of beta-secretase-like activity using a mass spectrometry-based assay system.

Gruninger-Leitch F, Berndt P, Langen H, Nelboeck P, Dobeli H.

CNS Research, Hoffmann-La Roche Ltd, Grenzacherstrasse 124, CH-4070 Basel, Switzerland.

We describe an assay system for the identification of site-specific proteases. The assay is based on a protein substrate that is immobilized on ceramic beads. After incubation with cell homogenates, the beads are washed and digested with endoproteinase Lys-C to liberate a defined set of peptides. The peptide fragments are identified by mass spectrometry. The assay was used to screen for beta-secretase, the protease that cleaves amyloid precursor protein (APP) at the beta-site. Cathepsin D was identified as the enzyme responsible for beta-secretase-like activity in two cell lines. Subsequent analysis of the related aspartic protease, cathepsin E, revealed almost identical cleavage specificity. Both enzymes are efficient in cleaving Swedish mutant APP at the beta-site but show almost no reactivity with wild-type APP. Treatment of cell lines with pepstatin inhibited the production of amyloid peptide (Abeta) when they were transfected with a construct bearing the Swedish APP mutant. However, when the cells were transfected with wild-type APP, the generation of Abeta was increased. This suggests that more than one enzyme is capable of generating Abeta in vivo and that an aspartic protease is involved in the processing of Swedish mutant APP.

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     Increased activity-regulating and neuroprotective efficacy
     .alpha.-secretase-derived secreted amyloid precursor protein conferred by
     a C-terminal heparin-binding domain
     Furukawa, Katsutoshi; Sopher, Bryce L.; Rydel, Russell E.; Begley, James
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      School and Center for Neurologic Diseases, Brigham and Women's Hospital,
      Boston, Massachusetts 02115, U.S.A.
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FS
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     The Genuine Article (R) Number: BT09W
     Toward the characterization and identification of gamma-secretases using
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     transition-state analogue inhibitors
     Moore C L; Diehl T S; Selkoe D J; Wolfe M S (Reprint)
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CS
     02115 USA (Reprint); Univ Tennessee, Dept Pharmaceut Sci, Memphis, TN
     38163 USA
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     Reference Count: 39
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     Catholic_Univ Louvain, Neuronal Cell Biol & Gene Transfer Lab, Ctr Human
     Genet, Flanders Interuniv Inst Biotechnol, Herestr 49, B-3000 Louvain,
     Belgium (Reprint); Catholic Univ Louvain, Neuronal Cell Biol & Gene
     Transfer Lab, Ctr Human Genet, Flanders Interuniv Inst Biotechnol, B-3000
     Louvain, Belgium; Univ Gottingen, Zentrum Biochem & Mol Zellbiol, D-3400
     Gottingen, Germany
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     *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*
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GΑ
     The Genuine Article (R) Number: 400MG
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                                ***beta***
     Amyloid precursor protein
                                               ***secretase***
                                                                    (BACE) mRNA
     expression in human neural cell lines following induction of neuronal
     differentiation and exposure to cytokines and growth factors
ΑU
     Satoh J (Reprint); Kuroda Y
CS
     Saga Med Sch, Dept Internal Med, Div Neurol, Saga 8498501, Japan
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     NEUROPATHOLOGY, ( ***DEC 2000*** ) vol. 20, No. 4, pp. 289-296.
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Protein kinase C regulation of intracellular and cell surface amyloid
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     precursor protein (APP) cleavage in CHO695 cells
     Jolly-Tornetta C; Wolf B A (Reprint)
ΑU
     Univ Penn, Sch Med, Dept Pathol & Lab Med, 230 John Morgan Bldg, 3620
CS
     Hamilton Walk, Philadelphia, PA 19104 USA (Reprint); Univ Penn, Sch Med,
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     The Genuine Article (R) Number: 380HX
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     from amyloid precursor protein overexpression in mice
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     UNIV MINNESOTA, SCH MED, DEPT NEUROL, CTR CLIN & MOL NEUROBIOL, BOX 295
     UMHC, 420 DELAWARE ST SE, MINNEAPOLIS, MN 55455 (Reprint); UNIV MINNESOTA.
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     2000:920752 SCISEARCH
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     The Genuine Article (R) Number: 378XT
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                     (BACE2) cleaves the amyloid precursor protein at the
        ***beta*** -
                       ***secretase*** site
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     Hussain I; Powell D J; Howlett D R; Chapman G A; Gilmour L; Murdock P R;
     Tew D G; Meek T D; Chapman C; Schneider K; Ratcliffe S J; Tattersall D;
     Testa T T; Southan C; Ryan D M; Simmons D L; Walsh F S; Dingwall C;
     Christie G (Reprint)
CS
     SMITHKLINE BEECHAM PHARMACEUT, DEPT NEUROSCI RES, NEW FRONTIERS SCI PK,
     HARLOW CM19 5AW, ESSEX, ENGLAND (Reprint); SMITHKLINE BEECHAM PHARMACEUT,
     DEPT NEUROSCI RES, HARLOW CM19 5AW, ESSEX, ENGLAND; SMITHKLINE BEECHAM
     PHARMACEUT, DEPT MOL SCREENING TECHNOL, HARLOW CM19 5AW, ESSEX, ENGLAND;
     SMITHKLINE BEECHAM PHARMACEUT, DEPT BIOTECHNOL & GENET, HARLOW CM19 5AW,
     ESSEX, ENGLAND; SMITHKLINE BEECHAM PHARMACEUT, DEPT DISCOVERY CHEM, HARLOW CM19 5AW, ESSEX, ENGLAND; SMITHKLINE BEECHAM PHARMACEUT, DEPT BIOINFORMAT,
     HARLOW CM19 5AW, ESSEX, ENGLAND; SMITHKLINE BEECHAM PHARMACEUT, DEPT MED CHEM, KING OF PRUSSIA, PA 19406; SMITHKLINE BEECHAM PHARMACEUT, DEPT MOL
     SCREENING TECHNOL, KING OF PRUSSIA, PA 19406
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     ENGLAND: USA
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     ANSWER 20 OF 116 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation.
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GΑ
     The Genuine Article (R) Number: 378XT
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     X11 alpha and X11 beta interact with presentlin-1 via their PDZ domains
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     Lau K F (Reprint); McLoughlin D M; Standen C; Miller C C J
     INST PSYCHIAT, DÉPT NEUROSCI, DENMARK HILL, LONDON SES 8AF, ENGLAND (Reprint); INST PSYCHIAT, OLD AGE PSYCHIAT SECT, LONDON SES 8AF, ENGLAND
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     Reference Count: 47
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AU CS 2000:797154 SCISEARCH

Nunan J; Small D H (Reprint)

The Genuine Article (R) Number: 365BZ

Regulation of APP cleavage by alpha-, beta- and gamma-secretases

UNIV MELBOURNE, DEPT PATHOL, MOL NEUROBIOL LAB, MELBOURNE, VIC 3010,

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AUSTRALIA (Reprint); UNIV MELBOURNE, DEPT PATHOL, MOL NEUROBIOL LAB,
      MELBOURNE, VIC 3010, AUSTRALIA
CYA
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      FEBS LETTERS, ( ***13 OCT 2000*** ) Vol. 483, No. 1, pp. 6-10.
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DT
      General Review; Journal
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      Reference Count: 51
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      The Genuine Article (R) Number: 365AA
      Coordinated expression of beta-amyloid precursor protein and the putative
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                     - ***secretase*** BACE and alpha-secretase ADAM10 in mouse
        ***beta***
      and human brain
      Marcinkiewicz M (Reprint); Seidah N G
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      CLIN RES INST MONTREAL, BIOCHEM NEUROENDOCRINOL LAB, 110 PINE AVE W.
      MONTREAL, PQ H2W 1R7, CANADA (Reprint)
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     The Genuine Article (R) Number: 363CB
     Proteolytic activation of recombinant pro-memapsin 2 (pro- ***beta*** - ***secretase*** ) studied with new fluorogenic substrates
Ermolieff J; Loy J A; Koelsch G; Tang J (Reprint)
OKLAHOMA MED RES FDN, PROT STUDIES PROGRAM, 825 NE 13TH ST, OKLAHOMA CITY,
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     OK 73104 (Reprint); OKLAHOMA MED RES FDN, PROT STUDIES PROGRAM, OKLAHOMA
      CITY, OK 73104; UNIV OKLAHOMA, HLTH SCI CTR, DEPT BIOCHEM & MOL BIOL,
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     Reference Count: 25
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AN
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     The Genuine Article (R) Number: 362DE
     Maturation and pro-peptide cleavage of
                                                   ***beta*** - ***secretase***
TT
ΑU
     Capell A; Steiner H; Willem M; Kaiser H; Meyer C; Walter J: Lammich S:
     Multhaup G; Haass C (Reprint)
CS
     UNIV MUNICH, ADOLF BUTENANDT INST, DEPT BIOCHEM, LAB ALZHEIMERS DIS RES,
     SCHILLERSTR 44, D-80336 MUNICH, GERMANY (Reprint); UNIV MUNICH, ADOLF
     BUTENANDT INST, DEPT BIOCHEM, LAB ALZHEIMERS DIS RES, D-80336 MUNICH, GERMANY; UNIV HEIDELBERG, CTR MOL BIOL ZMBH, D-69120 HEIDELBERG, GERMANY
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     PIKE, BETHESDA, MD 20814.
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secretase) complexed with inhibitor ***beta*** TI Hong L; Koelsch G; Lin X L; Wu S L; Terzyan S; Ghosh A K; Zhang X C; Tang J (Reprint) OKLAHOMA MED RES FDN, PROT STUDIES PROGRAM, 825 NE 13TH ST, OKLAHOMA CITY, CS OK 73104 (Reprint); OKLAHOMA MED RES FDN, PROT STUDIES PROGRAM, OKLAHOMA CITY, OK 73104; OKLAHOMA MED RES FDN, CRYSTALLOG PROGRAM, OKLAHOMA CITY, OK 73104; UNIV ILLINOIS, DEPT CHEM, CHICAGO, IL 60607; UNIV OKLAHOMA, HLTH SCI CTR, DEPT BIOCHEM & MOL BIOL, OKLAHOMA CITY, OK 73104 CYA USA SCIENCE, (***6 OCT 2000***) Vol. 290, No. 5489, pp. 150-153. SO Publisher: AMER ASSOC ADVANCEMENT SCIENCE, 1200 NEW YORK AVE, NW. WASHINGTON, DC 20005. ISSN: 0036-8075. DT Article; Journal PHYS; LIFE; AGRI FS English LA REC Reference Count: 32 *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS* ANSWER 29 OF 116 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN 2000:748990 SCISEARCH The Genuine Article (R) Number: 359FG GA Metabolism of amyloid precursor protein in COS cells transfected with a ***beta*** - ***secretase*** candidate Koike H; Kouchi Z; Kinouchi T; Maeda T; Sorimachi H; Saido T C; Maruyama K; Okuyama A; Suzuki K; Ishiura S (Reprint) CS UNIV TOKYO, GRAD SCH ARTS & SCI, DEPT LIFE SCI, MEGURO KU, 3-8-1 KOMABA, TOKYO 1538902, JAPAN (Reprint); UNIV TOKYO, GRAD SCH ARTS & SCI, DEPT LIFE SCI, MEGURO KU, TOKYO 1538902, JAPAN; UNIV TOKYO, INST MOL & CELLULAR BIOSCI, BUNKYO KU, TOKYO 1130032, JAPAN; TOKYO WOMENS MED UNIV, SCH MED, DEPT PHYSIOL, SHINJYUKU KU, TOKYO 1628666, JAPAN; JICHI MED SCH, DEPT BIOCHEM, MINAMI KAWACHI, TOCHIGI 3290498, JAPAN; UNIV TOKYO, GRAD SCH AGR & LIFE SCI, DEPT APPL BIOL CHEM, BIONKYO KOKYO 1130032, JAPAN; RIKEN, PRATIS AS A PROTECTIVE NEUROSCI, WAYO, SATTAMA 3510108 BRAIN SCI ÍNST, LAB PROTEOLYT NÉUROSCI, WAKO, SAITAMA 3510198, JÁPAN; TOKYO METROPOLITAN INST PSYCHIAT, DEPT MOL BIOL, SETAGAYA KU, 1568585, JAPAN; BANYU TSUKUBA RES INST, TSUKUBA, IBARAKI 3002611, JAPAN CYA **JAPAN** CYTOTECHNOLOGY. (***JUL 2000***) Vol. 33, No. 1-3, pp. 213-219. SO Publisher: KLUWER ACADEMIC PUBL, SPUIBOULEVARD 50, PO BOX 17, 3300 AA DORDRECHT, NETHERLANDS. ISSN: 0920-9069. Article; Journal AGRI FS LA English REC Reference Count: 21 *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS* ANSWER 30 OF 116 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN 2000:748036 SCISEARCH AN GA The Genuine Article (R) Number: 358VV TI Requirements for presenilin-dependent cleavage of notch and other transmembrane proteins ΑU Struhl G (Reprint); Adachi A COLUMBIA UNIV COLL PHYS & SURG, DEPT GENET & DEV, 701 W 168TH ST, NEW CS YORK, NY 10032 (Reprint); COLUMBIA UNIV COLL PHYS & SURG, HOWARD HUGHES MED INST, NEW YORK, NY 10032 CYA USA MOLECULAR CELL, (***SEP 2000***) Vol. 6, No. 3, pp. 625-636. SO Publisher: CELL PRESS, 1050 MASSACHUSETTES AVE, CIRCULATION DEPT, CAMBRIDGE, MA 02138. ISSN: 1097-2765. DT Article; Journal FS LIFE English LA REC Reference Count: 61 *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

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     GeorgeHyslop P H (Reprint); McLaurin J; Fraser P E
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     Remarkable roles of proteolysis on and beyond the cell surface
ΑU
     Blobel C P (Reprint)
     MEM SLOAN KETTERING CANC CTR, SLOAN KETTERING INST, CELLULAR BIOCHEM &
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     Dewachter I; VanDorpe J; Smeijers L; Gilis M; Kuiperi C; Laenen I;
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     FLEMISH INST BIOTECHNOL, CTR HUMAN GENET, EXPT GENET GRP, B-3000 LOUVAIN,
     BELGIUM; CNRS, INST PHARMACOL MOL & CELLULAIRE, UNITE PROPRE RECH 411,
     F-06560 VALBONNE, FRANCE; JANSSEN RES FDN, B-2340 BEERSE, BELGIUM;
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     The Genuine Article (R) Number: 346VF
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     Isoform-specific increases following heat shock
     Shepherd C E; Bowes S; Parkinson D; CambrayDeakin M; Pearson R C A
     (Reprint)
     UNIV SHEFFIELD, DEPT BIOMED SCI, WESTERN BANK, SHEFFIELD S10 2TN, S
CS
     YORKSHIRE, ENGLAND (Reprint); UNIV SHEFFIELD, DEPT BIOMED SCI, SHEFFIELD
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\$10 2TN, S YORKSHIRE, ENGLAND; SHEFFIELD HALLAM UNIV, DEPT BIOMED SCI,

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     Reference Count: 50
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     2000:644266 SCISEARCH
     The Genuine Article (R) Number: 345XM
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               ***beta*** - ***secretase***
TI
                                                   homolog, cleaves at the beta
     site and within the amyloid-beta region of the amyloid-beta precursor
     protein
     Farzan M (Reprint); Schnitzler C E; Vasilieva N; Leung D; Choe H
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     DANA FARBER CANC INST, DEPT CANC IMMUNOL & AIDS, BOSTON, MA 02115
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     (Reprint); CHILDRENS HOSP, DEPT PATHOL, BOSTON, MA 02115; CHILDRENS HOSP,
     PERLMUTTER LAB, BOSTON, MA 02115; HARVARD UNIV, SCH MED, DEPT MED, BOSTON,
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     The Genuine Article (R) Number: 332QG
GA
TI
     Expression analysis of BACE2 in brain and peripheral tissues
ΑU
     Bennett B D; BabuKhan S; Loeloff R; Louis J C; Curran E; Citron M; Vassar
     R (Reprint)
     AMGEN INC, 1 AMGEN CTR DR, M-S 29-2-B, THOUSAND OAKS, CA 91320 (Reprint); AMGEN INC, THOUSAND OAKS, CA 91320
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     The Genuine Article (R) Number: 329QF
TT
     Regulation of amyloid precursor protein (APP) secretion by protein kinase
     C alpha in human ntera 2 neurons (NT2N)
     JollyTornetta C; Wolf B A (Reprint)
CS
     UNIV PENN, SCH MED, DEPT PATHOL & LAB MED, 230 JOHN MORGAN BLDG, 3260
     HAMILTON WALK, PHILADELPHIA, PA 19104 (Reprint); UNIV PENN, SCH MED, DEPT
     PATHOL & LAB MED, PHILADELPHIA, PA 19104
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     The Genuine Article (R) Number: 328WF
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     A yeast genetic assay for caspase cleavage of the amyloid-beta precursor
     Gunyuzlu P L (Reprint); White W H; Davis G L; Hollis G F; Toyn J H
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     DUPONT PHARMACEUT, EXPT STN, WILMINGTON, DE 19880
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     The Genuine Article (R) Number: 317ZA
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     protein precursor transgenic mice: Synaptotoxicity without plaque
     formation
     Mucke L (Reprint); Masliah E; Yu G Q; Mallory M; Rockenstein E M; Tatsuno
     G; Hu K; Kholodenko D; Johnsonwood K; McConlogue L
CS
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     The Genuine Article (R) Number: 319BG
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     Presenilin 1 is linked with gamma-secretase activity in the detergent
     solubilized state
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     Li Y M (Reprint); Lai M T; Xu M; Huang Q; DiMuzioMower J; Sardana M K; Shi
     X P; Yin K C; Shafer J A; Gardell S J
    MERCK RES LABS, DEPT BIOL CHEM, W POINT, PA 19486 (Reprint)
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      *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*
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AN
      2000:166635 SCISEARCH
      The Genuine Article (R) Number: 285VU
GA
      Human aspartic protease memapsin 2 cleaves the ***secretase*** site of beta-amyloid precur
                                                                ***beta***
TT
                            site of beta-amyloid precursor protein
      Lin X L; Koelsch C; Wu S L; Downs D; Dashti A; Tang J (Reprint)
OKLAHOMA MED RES FDN, PROT STUDIES PROGRAM, 825 NE 13TH ST, OKLAHOMA CITY,
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CS
      OK 73104 (Reprint); OKLAHOMA MED RES FDN, PROT STUDIES PROGRAM, OKLAHOMA
      CITY, OK 73104; UNIV OKLAHOMA, HLTH SCI CTR, DEPT BIOCHEM & MOL BIOL,
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      The Genuine Article (R) Number: 286PA
GΑ
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      The gene encoding DRAP (BACE2), a glycosylated transmembrane protein of
      the aspartic protease family, maps to the Down critical region
      Acquati_F; Accarino M; Nucci C; Fumagalli P; Jovine L; Ottolenghi S:
      Taramelli R (Reprint)
      UNIV INSUBRIA, DIPARTIMENTO STRUTTURALE & FUNZIONALE, VIA DUNANT 3, VARESE, ITALY (Reprint); UNIV INSUBRIA, DIPARTIMENTO STRUTTURALE &
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      The Genuine Article (R) Number: 284ME
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      cells expressing mutants of the amyloid precursor protein defective in
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     Cescato R; Dumermuth E; Spiess M; Paganetti P A (Reprint)
NOVARTIS PHARMA AG, POB WSJ 386-8-28, CH-4002 BASEL, SWITZERLAND
(Reprint); NOVARTIS PHARMA AG, CH-4002 BASEL, SWITZERLAND; UNIV BASEL,
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GΑ
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                                                           ***beta***
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       ***secretase***
                          cleavage site
     Abbenante G (Reprint); Kovacs D M; Leung D L; Craik D J; Tanzi R E;
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     Fairlie D P
     UNIV QUEENSLAND, CTR DRUG DESIGN & DEV, BRISBANE, QLD 4072, AUSTRALIA (Reprint); HARVARD UNIV, MASSACHUSETTS GEN HOSP, SCH MED, DEPT NEUROL,
CS
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     2000:114055 SCISEARCH
     The Genuine Article (R) Number: 280XA
GA
TI
     Protein kinase C-dependent alpha-secretase competes with
                                                                   ***beta***
       ***secretase***
                          for cleavage of amyloid-beta precursor protein in the
     trans-Golgi network
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     Skovronsky D M; Moore D B; Milla M E; Doms R W; Lee V M Y (Reprint)
     HUP, CTR NEURODEGENERAT DIS RES, DEPT PATHOL & LAB MED, MALONEY 3.
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     The Genuine Article (R) Number: 278LX
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     Presentlin I expression in yeast lowers secretion of the amyloid precursor
     protein
     Evin G (Reprint); LeBrocque D; Culvenor J G; Galatis D; Weidemann A;
     Beyreuther K; Masters C L; Cappai R
CS
     UNIV MELBOURNE, DEPT PATHOL, PARKVILLE, VIC 3052, AUSTRALIA (Reprint);
     MENTAL HLTH RES INST, PARKVILLE, VIC 3052, AUSTRALIA; UNIV HEIDELBERG,
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The Genuine Article (R) Number: 278LX

GΑ

beta - ***secretase*** -like activity for TI The 68K protease has lymphocyte precursor protein but not for brain substrate Matsumoto A (Reprint) KOBE UNIV, SCH MED, DEPT RADIAT BIOPHYS & GENET, KUSUNOKI CHO 7, KOBE, CS HYOGO 6500017, JAPAN (Reprint) CYA **JAPAN** NEUROREPORT. (***7 FEB 2000***) Vol. 11, No. 2, pp. 373-377. SO Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621. ISSN: 0959-4965. DT Article; Journal LIFE FS English LA REC Reference Count: 19 *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS* L4 ANSWER 49 OF 116 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN ΑN 2000:57987 SCISEARCH The Genuine Article (R) Number: 273GQ Identification of ***beta*** - ***secretase*** -like activity using a GΑ TI mass spectrometry-based assay system GruningerLeitch F; Berndt P; Langen H; Nelboeck P; Dobeli H (Reprint) ΑU CS F HOFFMANN LA ROCHE & CO LTD, CNS RES, GRENZACHERSTR 124, CH-4070 BASEL, SWITZERLAND (Reprint); F HOFFMANN LA ROCHE & CO LTD, CNS RES, CH-4070 BASEL, SWITZERLAND; F HOFFMANN LA ROCHE & CO LTD, ROCHE GENET, CH-4070 BASEL, SWITZERLAND CYA **SWITZERLAND** NATURE BIOTECHNOLOGY, (***JAN 2000***) Vol. 18, No. 1, pp. 66-70. SO. Publisher: NATURE AMERICA INC, 345 PARK AVE SOUTH, NEW YORK, NY 10010-1707 ISSN: 1087-0156. Article; Journal DT FS LIFE; AGRI LA English REC Reference Count: 23 *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS* L4 ANSWER 50 OF 116 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN 2000:48562 SCISEARCH The Genuine Article (R) Number: 272RT The function of presentlin-1 in amyloid beta-peptide generation and brain TI development ΑU Saftig P (Reprint); Hartmann D; DeStrooper P UNIV GOTTINGEN, ZENTRUM BIOCHEM & MOL ZELLBIOL, BIOCHEM ABT 2, CS HEINRICH-DUKER-WEG 12, D-37027 GOTTINGEN, GERMANY (Reprint); CHRISTIAN ALBRECHTS UNIV KIEL, INST ANAT, D-24098 KIEL, GERMANY; KATHOLIEKE UNIV LEUVEN, CTR HUMAN GENET, FLANDERS INST BIOTECHNOL VIB4, LOUVAIN, BELGIUM GERMANY; BELGIUM CYA EUROPEAN ARCHIVES OF PSYCHIATRY AND CLINICAL NEUROSCIENCE, (***DEC*** 1999***) Vol. 249, No. 6, pp. 271-279. Publisher: DR DIETRICH STEINKOPFF VERLAG, PLATZ DER DEUTSCHEN EINHEIT 25, D-64293 DARMSTADT, GERMANY. ISSN: 0940-1334 Article; Journal DT FS LIFE; CLIN LA English REC Reference Count: 87 *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS* L4 ANSWER 51 OF 116 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN 2000:25182 SCISEARCH The Genuine Article (R) Number: 269RD TI Identification of a novel aspartic protease (Asp 2) as ***beta*** ***secretase*** Hussain I; Powell D; Howlett D R; Tew D G; Week T D; Chapman C; Gloger I S; Murphy K E; Southan C D; Ryan D M; Smith T S; Simmons D L; Walsh F S; Dingwall C (Reprint); Christie G CS SMITHKLINE BEECHAM PHARMACEUT, DEPT NEUROSCI, NEW FRONTIERS SCI PK N. COLDHARBOUR RD, 4TH AVE, HARLOW CM19 5AW, ESSEX, ENGLAND (Reprint); SMITHKLINE BEECHAM PHARMACEUT, DEPT NEUROSCI, HARLOW CM19 5AW, ESSEX, ENGLAND; SMITHKLINE BEECHAM PHARMACEUT, DEPT MOL SCREENING TECHNOL, HARLOW CM19 5AW, ESSEX, ENGLAND; SMITHKLINE BEECHAM PHARMACEUT, DEPT BIOTECHNOL & GENET, HARLOW CM19 5AW, ESSEX, ENGLAND; SMITHKLINE BEECHAM PHARMACEUT,

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     JAPAN; YOKOHAMA CITY UNIV, SCH MED, DEPT BIOL MOL, KANAZAWA KU, KANAGAWA
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     Sinha S (Reprint); Lieberburg I
     ELAN PHARMACEUT, S SAN FRANCISCO, CA 94080 (Reprint)
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     Frears E R; Stephens D J; Walters C E; Davies H; Austen B M (Reprint)
     UNIV LONDON ST GEORGES HOSP, SCH MED, NEURODEGENERAT UNIT, CRANMER
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     SCH MED, NEURODEGENERAT UNIT, LONDON SW17 ORE, ENGLAND; CIPHERGEN BIOSYST
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                                                     ***beta*** - ***secretase***
     -like activity
     Abraham C R; Marshall D C L; Tibbles H E; Otto K; Long H J; Billingslea A M; Hastey R; Johnson R; Fine R E; Smith S J; Simons E R; Davies T A
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     BOSTON UNIV, SCH MED, 80 E CONCORD ST, K6, BOSTON, MA 02118 (Reprint):
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     ISSN: 0022-2143.
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     The Genuine Article (R) Number: 188EX
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     gamma-Secretase, evidence for multiple proteolytic activities and
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     beta peptides of varying length
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     Murphy M P; Hickman L J; Eckman C B; Uljon S N; Wang R; Golde T E
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CS
     MAYO CLIN JACKSONVILLE, DEPT PHARMACOL, 4500 SAN PABLO RD, JACKSONVILLE,
     FL 32224 (Reprint); MAYO CLIN JACKSONVILLE, DEPT PHARMACOL, JACKSONVILLE,
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     UNIV, LAB MASS SPECTROMETRY, NEW YORK, NY 10021
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     Pulse-chase experiments revealed
     cleavage from immature full-length amyloid precursor protein harboring the
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     Urmoneit B (Reprint); Turner J; Dyrks T
ΑU
CS
     UNIV DUSSELDORF, MOORENSTR 5, D-40225 DUSSELDORF, GERMANY (Reprint);
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     Greengard P; Relkin N R; Gandy S
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     10021 (Reprint); ROCKEFELLER UNIV, MOL & CELLULAR NEUROSCI LAB, NEW YORK.
     NY 10021; ROCKEFELLER UNIV, LAB MASS SPECTROMETRY, NEW YORK, NY 10021;
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     Davies T A; Billingslea A M; Long H J; Tibbles H; Wells J M; Eisenhauer P
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     Stephens D J (Reprint); Austen B M
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     Lack of beta-amyloid production in M19 cells deficient in site 2
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     Manni M E; Cescato R; Paganetti P A (Reprint)
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     NOVARTIS PHARMA LTD, CH-4002 BASEL, SWITZERLAND (Reprint); NOVARTIS PHARMA
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     apoptosis
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     Hypothesis: beta amyloid precursor protein is a key sorting and targeting
     receptor for neuropeptidases
     Fine R E (Reprint); Abraham C R
     BOSTON UNIV, SCH MED, DEPT BIOCHEM, K-124C, 80 E CONCORD ST, BOSTON, MA 02118 (Reprint); BOSTON UNIV, SCH MED, DEPT MED, BOSTON, MA 02118; EDITH
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      (matrix metalloproteinase 2)-deficient mice
      Itoh T; Ikeda T; Gomi H; Nakao S; Suzuki T; Itohara S (Reprint) KYOTO UNIV, INST VIRUS RES, SAKYO KU, 53 KAWAHARA, KYOTO 60601, JAPAN (Reprint); KYOTO UNIV, INST VIRUS RES, SAKYO KU, KYOTO 60601, JAPAN; UNIV TOKYO, FAC PHARMACEUT SCI, BUNKYO KU, TOKYO 113, JAPAN
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Novel ***beta*** - ***secretase***
GA
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      NT2N cells
      Chyung A S C; Greenberg B D; Cook D G; Doms R W; Lee V M Y (Reprint)
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      The Genuine Article (R) Number: XA406
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      Examination of the role of endopeptidase 3.4.24.15 in A beta secretion by
      human transfected cells
      Chevallier N; Jiracek J; Vincent B; Baur C P; Spillantini M G; Goedert M;
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      Dive V; Checler F (Reprint)
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MAYO CLIN JACKSONVILLE, 4500 SAN PABLO RD, JACKSONVILLE, FL 32224

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     RESERVE UNIV, DEPT NEUROSCI, CLEVELAND, OH 44106; UNIV ALABAMA, DEPT
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     AMYLOID PRECURSOR PROTEIN TRUNCATED AT ANY OF THE GAMMA-SECRETASE SITES IS
     NOT CLEAVED TO BETA-AMYLOID
     PAGANETTI P A (Reprint); LIS M; KLAFKI H W; STAUFENBIEL M
CS
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GΑ
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                                         LOCALIZE TO THE TRANS-GOLGI NETWORK AND
     LATE ENDOSOME IN 293 CELLS
     STEPHENS D J; AUSTEN B M (Reprint)
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     ST GEORGE HOSP, SCH MED, DEPT SURG, CRANMER TERRACE, LONDON SW17 ORE
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     SUBSTITUTION OF PHENYLALANINE BY PROLINE AT POSITION-19 OF AMYLOID-BETA
     PEPTIDE RESULTS IN AN INCREASED PRODUCTION OF AMYLOID-BETA PEPTIDES WITH
     ALTERNATIVE N-TERMINI AFTER PROTEIN-KINASE-C STIMULATION
     CAPELL A; TEPLOW D B; CITRON M; SELKOE D J; HAASS C (Reprint)
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     UNIV HEIDELBERG, CENT INST MENTAL HLTH, DEPT MOL BIOL, D-68195 MANNHEIM,
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     MA, 02115; BRIGHAM & WOMENS HOSP, BIOPOLYMER LAB, BOSTON, MA, 02115;
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- REAUME A G; HOWLAND D S; TRUSKO S P; SAVAGE M J; LANG D M; GREENBERG B D; SIMAN R; SCOTT R W (Reprint)
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- KUENTZEL S L; GONZALEZDEWHITT P A; LOWERY D E; ALTMAN R A; LEONE J W; HEINRIKSON R L; GREENBERG B D; RAUB T J (Reprint)
- UPJOHN CO, UPJOHN LABS, DRUG DELIVERY SYST RES, KALAMAZOO, MI, 49001 (Reprint); UPJOHN CO, UPJOHN LABS, DRUG DELIVERY SYST RES, KALAMAZOO, MI, CS 49001; UPJOHN CO, UPJOHN LABS, MOLEC BIOL RES, KALAMAZOO, MI, 49001; UPJOHN CO, UPJOHN LABS, CENT NERVOUS SYST RES, KALAMAZOO, MI, 49001; UPJOHN CO, UPJOHN LABS, BIOCHEM, KALAMAZOO, MI, 49001; CEPHALON INC, W CHESTER, PA, 19380; UPJOHN CO, UPJOHN LABS, ANIM HLTH DISCOVERY RES KALAMAZOO, MI, 49001; MILES INC, DIV PHARMACEUT, INST MOLEC BIOL, WEST HAVEN, CT, 06516
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- ***BETA*** ***SECRETASE*** TI HUMAN BRAIN CONTAINS HEPARAN-SULFATE

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     650, JAPAN (Reprint); OSAKA TEISHIN HOSP, DEPT INTERNAL MED 1, OSAKA 543,
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***BETA*** - ***SECRETASE*** PROTEASES USING PEPTIDE AND AMYLOID
TT
                      ***SECRETASE***
                                         PROTEASES USING PEPTIDE AND AMYLOID
     PRECURSOR PROTEIN SUBSTRATES
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     (Reprint)
     WYETH AYERST RES, CNS DISORDERS, PRINCETON, NJ, 08543 (Reprint); WYETH
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                        ***15 APR 1996*** ) Vol. 716, No. 1-2, pp. 91-100.
SO
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DT
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FS
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     Reference Count: 44
     *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*
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     96:359561 SCISEARCH
GΑ
     The Genuine Article (R) Number: BF30Z
     APP GENE FAMILY - ALTERNATIVE SPLICING GENERATES FUNCTIONALLY RELATED
     ISOFORMS
     SANDBRINK R (Reprint); MASTERS C L; BEYREUTHER K
     UNIV HEIDELBERG, ZENTRUM MOLEK BIOL HEIDELBERG, NEUENHEIMER FELD 282,
CS
     D-69120 HEIDELBERG, GERMANY (Reprint); UNIV MELBOURNE, DEPT PATHOL,
     PARKVILLE, VIC 3052, AUSTRALIA
CYA
     GERMANY; AUSTRALIA
     ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, ( ***1996*** ) Vol. 777, pp.
SO.
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     ISSN: 0077-8923.
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     Reference Count: 19
     *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*
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     ANSWER 91 OF 116 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation.
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GLYCOCONJUGATES

on STN

- 96:359529 SCISEARCH AN The Genuine Article (R) Number: BF30Z GA THE ROLE OF APP PROCESSING AND TRAFFICKING PATHWAYS IN THE FORMATION OF AMYLOID BETA-PROTEIN SELKOE D J (Reprint); YAMAZAKI T; CITRON M; PODLISNY M B; KOO E H; TEPLOW D B; HAASS C CS HARVARD UNIV, BRIGHAM & WOMENS HOSP, SCH MED, CTR NEUROL, DEPT NEUROL, 221 LONGWOOD AVE, BOSTON, MA, 02115 (Reprint) CYA SO ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (***1996***) Vol. 777, pp. 57-64. ISSN: 0077-8923. DT Article; Journal FS LIFE LA **ENGLISH** REC Reference Count: 19 *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS* 14 ANSWER 92 OF 116 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN 96:303945 SCISEARCH ΑN The Genuine Article (R) Number: UE730 GA TT ENHANCED RELEASE OF AMYLOID BETA-PROTEIN FROM CODON-670/671 SWEDISH MUTANT BETA-AMYLOID PRECURSOR PROTEIN OCCURS IN BOTH SECRETORY AND ENDOCYTIC **PATHWAYS** ΑU PEREZ R G; SQUAZZO S L; KOO E H (Reprint) CS HARVARD UNIV, BRIGHAM & WOMENS HOSP, SCH MED, CTR NEUROL DIS, 221 LONGWOOD AVE, LMRC 114, BOSTON, MA, 02115 (Reprint); HARVARD UNIV, BRIGHAM & WOMENS HOSP, SCH MED, CTR NEUROL DIS, BOSTON, MA, 02115; HARVARD UNIV, BRIGHAM & WOMENS HOSP, SCH MED, DEPT NEUROL, BOSTON, MA, 02115; HARVARD UNIV, BRIGHAM & WOMENS HOSP, SCH MED, DEPT PATHOL, BOSTON, MA, 02115 CYA JOURNAL OF BIOLOGICAL CHEMISTRY, (***12 APR 1996***) Vol. 271, No. 15, SO pp. 9100-9107 ISSN: 0021-9258. DT Article; Journal FS LIFE **ENGLISH** LA REC Reference Count: 42 *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS* L4 ANSWER 93 OF 116 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN 96:303429 SCISEARCH AN The Genuine Article (R) Number: UE776 GA MONOCLONAL-ANTIBODIES AGAINST THE HUMAN METALLOPROTEASE EC-3.4.24.15 LABEL NEUROFIBRILLARY TANGLES IN ***ALZHEIMERS*** - ***DISEASE*** BRAIN TI ΑU CONN K J; PIETROPAOLO M; JU S T; ABRAHAM C R (Reprint) CS BOSTON UNIV, SCH MED, CTR ARTHRITIS, DEPT MED, K-5, 80 E CONCORD ST, BOSTON, MA, 02118 (Reprint); BOSTON UNIV, SCH MED, CTR ARTHRITIS, DEPT MED, BOSTON, MA, 02118; BOSTON UNIV, SCH MED, DEPT BIOCHEM, BOSTON, MA, 02118; BOSTON UNIV, SCH MED, DEPT PATHOL, BOSTON, MA. 02118 CYA JOURNAL OF NEUROCHEMISTRY, (***MAY 1996***) Vol. 66, No. 5, pp. SO. 2011-2018 ISSN: 0022-3042. DT Article: Journal FS LIFE
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- REC Reference Count: 46 *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*
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- The Genuine Article (R) Number: TQ573
- AMYLOIDOGENIC PROCESSING OF THE HUMAN AMYLOID PRECURSOR PROTEIN IN PRIMARY CULTURES OF RAT HIPPOCAMPAL-NEURONS
- SIMONS M; DESTROOPER B; MULTHAUP G; TIENARI P J; DOTTI C G; BEYREUTHER K (Reprint)
- CS UNIV HEIDELBERG, CTR MOLEC BIOL HEIDELBERG ZMBH, NEUENHEIMER FELD 282, D-69120 HEIDELBERG, GERMANY (Reprint); UNIV HEIDELBERG, CTR MOLEC BIOL HEIDELBERG ZMBH, D-69120 HEIDELBERG, GERMANY; EUROPEAN MOLEC BIOL LAB, CELL BIOL PROGRAM, D-69012 HEIDELBERG, GERMANY; CATHOLIC UNIV LEUVEN, CTR HUMAN GENET, B-3000 LOUVAIN, BELGIUM
- CYA GERMANY; BELGIUM

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GA
     The Genuine Article (R) Number: TP754
     INTRACELLULAR GENERATION OF AMYLOID BETA-PROTEIN FROM AMYLOID BETA-PROTEIN
     PRECURSOR FRAGMENT BY DIRECT CLEAVAGE WITH
                                                       ***BETA***
        ***SECRETASE***
                           AND GAMMA-SECRETASE
     IIZUKA T; SHOJI M (Reprint); KAWARABAYASHI T; SATO M; KOBAYASHI T; TADA N; KASAI K; MATSUBARA E; WATANABE M; TOMIDOKORO Y; HIRAI S GUNMA UNIV, SCH MED, DEPT NEUROL, 3-39-15 SHOWA MACHI, MAEBASHI, GUMMA 371, JAPAN (Reprint); GUNMA UNIV, SCH MED, DEPT NEUROL, MAEBASHI, GUMMA
CS
     371, JAPAN; HOECHST JAPAN LTD, PHARMA RES & DEV DIV, LAB ANIM CTR,
     KAWAGOE, SAITAMA 35011, JAPAN
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     The Genuine Article (R) Number: TM328
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                                 ***BETA***
                                                - ***SECRETASE*** -LIKE ACTIVITY
TT
     GELATINASE-A POSSESSES A
     IN CLEAVING THE AMYLOID PROTEIN-PRECURSOR OF
                                                         ***ALZHEIMERS***
        ***DISEASE***
     LEPAGE R N; FOSANG A J; FULLER S J; MURPHY G; EVIN G; BEYREUTHER K; MASTERS C L; SMALL D H (Reprint)
     UNIV MELBOURNE, DEPT PATHOL, MELBOURNE NERVE GROWTH RES UNIT, MOLEC
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     PARKVILLE, VIC 3052, AUSTRALIA; UNIV MELBOURNE, DEPT PATHOL, PARKVILLE,
     VIC 3052, AUSTRALIA; UNIV MELBOURNE, DEPT PAEDIAT, ORTHOPAED MOLEC BIOL
     RES UNIT, PARKVILLE, VIC 3052, AUSTRALIA; STRANGEWAYS RES LAB, CAMBRIDGE
     CB1 4RN, ENGLAND; UNIV HEIDELBERG, CTR MOLEC BIOL, W-6900 HEIDELBERG,
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     The Genuine Article (R) Number: TH786
GΑ
     THE SWEDISH MUTATION CAUSES EARLY-ONSET
                                                  ***ALZHEIMERS***
       ***DISEASE***
                                              ***SECRETASE***
                              ***BETA*** -
                                                                 CLEAVAGE WITHIN THE
                        BY
     SECRETORY PATHWAY
ΑU
     HAASS C (Reprint); LEMERE C A; CAPELL A; CITRON M; SEUBERT P; SCHENK D;
     LANNFELT L; SELKOÉ D J
     HARVARD UNIV, BRIGHAM & WOMENS HOSP, SCH MED, CTR NEUROL DIS, BOSTON, MA,
CS
     02115 (Reprint); UNIV HEIDELBERG, CENT INST MENTAL HLTH, DEPT MOLEC BIOL,
     D-68159 MANNHEIM, GERMANY; ATHENA NEUROSCI INC, S SAN FRANCISCO, CA
     94080; KAROLINSKA INST, DEPT GERIATR MED 856, S-14186 HUDDINGE, SWEDEN
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     USA; GERMANY; SWEDEN
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AN
      The Genuine Article (R) Number: TA741
GΑ
      INHIBITION OF BETA-A4 PRODUCTION BY SPECIFIC MODULATION OF
TI
         ***SECRETASE***
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ΑU
      URMONEIT B; REINSCH C; TURNER J (Reprint); CZECH C; BEYREUTHER K; DYRKS T
      SCHERING AG, RES LABS, D-13342 BERLIN, GERMANY (Reprint); SCHERING AG, RES
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      The Genuine Article (R) Number: TA184
GΑ
      BRAIN CATHEPSIN-B BUT NOT METALLOENDOPEPTIDASES DEGRADE RAPP(751) WITH
TT
      PRODUCTION OF AMYLOIDOGENIC FRAGMENTS - COMPARISON WITH SYNTHETIC PEPTIDES EMULATING ***BETA*** - ***SECRETASE*** AND GAMMA-SECRETASE SITES
      MARKS N (Reprint); BERG M J; SAPIRSTEIN V S; DURRIE R; SWISTOK J; MAKOFSKE
AU
      R C; DANHO W
CS
      NATHAN S KLINE INST PSYCHIAT RES, DEPT NEUROCHEM, 140 OLD ORANGEBURG RD,
      ORANGEBURG, NY, 10962 (Reprint); NATHAN S KLINE INST PSYCHIAT RES, DEPT
      NEUROBIOL, ORANGEBURG, NY, 10962; NYU, SCH MED, DEPT PSYCHIAT, NUTLEY, NJ,
      00000; ROCHE RES CTR, NUTLEY, NJ, 00000
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L4
      on STN
      95:555710 SCISEARCH
AN
GΑ
      The Genuine Article (R) Number: RP189
                                         ***ALZHEIMERS*** - ***DISEASE***
ΤI
      AMYLOID-BETA AMYLOIDOSIS IN
     PRICE D L (Reprint); SISODIA S S; GANDY S E
JOHNS HOPKINS UNIV, SCH MED, NEUROPATHOL LAB, 558 ROSS RES BLDG, 720
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NEUROL, BALTIMORE, MD, 21205; JOHNS HOPKINS UNIV, SCH MED, DEPT NEUROSCI,
BALTIMORE, MD, 21205; CORNELL UNIV, MED CTR, NEW YORK HOSP, DEPT NEUROSCI,
NEW YORK NY 10021: CORNELL UNIV, MED CTR, NEW YORK HOSP, DEPT NEUROSCI
CS
      NEW YORK, NY, 10021; CORNELL UNIV, MED CTR, NEW YORK HOSP, DEPT NEUROSCI,
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      95:520955 SCISEARCH
AN
      The Genuine Article (R) Number: RL488
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TI

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     SCOTT R W
CS
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     95:315697
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     The Genuine Article (R) Number: QW601
TT
     THE RAS-RELATED GTP-BINDING PROTEIN, RAB1B, REGULATES EARLY STEPS IN
     EXOCYTIC TRANSPORT AND PROCESSING OF BETA-AMYLOID PRECURSOR PROTEIN
     DUGAN J M; DEWIT C; MCCONLOGUE L; MALTESE W A (Reprint)
AU
CS
     WEIS CTR RES, GEISINGER CLIN, 100 N ACAD AVE, DANVILLE, PA, 17822
     (Reprint); WEIS CTR RES, GEISINGER CLIN, DANVILLE, PA, 17822; ATHENA
     NEUROSCI, S SAN FRANCISCO, CA, 94080
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     USA
     JOURNAL OF BIOLOGICAL CHEMISTRY, ( ***05 MAY 1995*** ) Vol. 270, No. 18.
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     pp. 10982-10989.
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     Reference Count: 73
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     The Genuine Article (R) Number: QP451
     ROLE OF THE BETA-AMYLOID PROTEIN IN
                                             ***ALZHEIMERS*** - ***DISEASE***
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     SISODIA S S (Reprint); PRICE D L
     JOHNS HOPKINS UNIV, SCH MED, NEUROPATHOL LAB, 558 ROSS RES BLDG, 720
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     RUTLAND AVE, BALTIMORE, MD, 21205 (Reprint); JOHNS HOPKINS UNIV, SCH MED,
     DEPT PATHOL, BALTIMORE, MD, 21205; JOHNS HOPKINS UNIV, SCH MED, DEPT
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     Reference Count: 77
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     on STN
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GΑ
     The Genuine Article (R) Number: QP230
TI
     GENERATION OF AMYLOID-BETA PROTEIN FROM ITS PRECURSOR IS SEQUENCE-SPECIFIC
     CITRON M (Reprint); TEPLOW D B; SELKOE D J
ΑU
CS
     HARVARD UNIV, BRIGHAM & WOMENS HOSP, SCH MED, CTR NEUROL DIS, DEPT NEUROL,
     PROGRAM NEUROSCI, BOSTON, MA, 02115 (Reprint)
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     Reference Count: 34
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- GΑ The Genuine Article (R) Number: QH688
- BASOLATERAL SECRETION OF AMYLOID PRECURSOR PROTEIN IN MADIN-DARBY CANINE TI KIDNEY-CELLS IS DISTURBED BY ALTERATIONS OF INTRACELLULAR PH AND BY INTRODUCING A MUTATION ASSOCIATED WITH FAMILIAL ***ALZHEIMERS*** ***DISEASE***
- DESTROOPER B; CRAESSAERTS K; DEWACHTER I; MOECHARS D; GREENBERG B; ΑU VANLEUVEN F (Reprint); VANDENBERGHE H
- KATHOLIEKE UNIV LEUVEN, CTR HUMAN GENET, DEPT HUMAN GENET, EXPTL GENET GRP, B-3000 LOUVAIN, BELGIUM (Reprint); KATHOLIEKE UNIV LEUVEN, CTR HUMAN CS GENET, DEPT HUMAN GENET, EXPTL GENET GRP, B-3000 LOUVAIN, BELGIUM; CEPHALON INC, W CHESTER, PA, 19380
- CYA BELGIUM; USA
- JOURNAL OF BIOLOGICAL CHEMISTRY, (***24 FEB 1995***) Vol. 270. No. 8. SO pp. 4058-4065. ISSN: 0021-9258
- DT Article; Journal
- FS LIFE
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- REC Reference Count: 52 *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*
- ANSWER 106 OF 116 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
- 95:122625 SCISEARCH AΝ
- GΑ The Genuine Article (R) Number: QF535
- TI PROTEIN-TYPE SPECIFIC-INHIBITION OF A-BETA RELEASE BY BAFILOMYCIN A1. A SELECTIVE INHIBITOR OF VACUOLAR ATPASES
- KNOPS J; SUOMENSAARI S; LEE M; MCCONLOGUE L; SEUBERT P; SINHA S (Reprint) ATHENA NEUROSCI INC, S SAN FRANCISCO, CA, 94080 (Reprint); ATHENA NEUROSCI CS INC, S SAN FRANCISCO, CA, 94080
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- DT Note; Journal
- FS LIFE
- LA **ENGLISH**
- REC Reference Count: 26 *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*
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- 95:93527 SCISEARCH
- The Genuine Article (R) Number: QC813
 ALZHEIMERS ***DISEASE*** TT AMYLOID PRECURSOR PROTEIN (A-BETA-PP) - PROTEOLYTIC PROCESSING, SECRETASES AND BETA-A4 AMYLOID **PRODUCTION**
- AU EVIN G; BEYREUTHER K; MASTERS C L (Reprint)
- UNIV MELBOURNE, DEPT PATHOL, PARKVILLE, VIC 3052, AUSTRALIA (Reprint); CS UNIV MELBOURNE, DEPT PATHOL, PARKVILLE, VIC 3052, AUSTRALIA; MENTAL HLTH RES INST, PARKVILLE, VIC 3052, AUSTRALIA; UNIV HEIDELBERG, CTR MOLEC BIOL, HEIDELBERG, VIC, AUSTRALIA
- CYA **AUSTRALIA**
- SO AMYLOID-INTERNATIONAL JOURNAL OF EXPERIMENTAL AND CLINICAL INVESTIGATION, ***DEC 1994***) Vol. 1, No. 4, pp. 263-280. ISSN: 1350-6129.
- DT General Review; Journal
- FS LIFE
- **ENGLISH** LA
- REC Reference Count: 143 *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*
- L4 ANSWER 108 OF 116 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
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- The Genuine Article (R) Number: PV510
- TT METABOLISM OF THE SWEDISH AMYLOID PRECURSOR PROTEIN VARIANT IN MADIN-DARBY CANINE KIDNEY-CELLS
- LO A C Y; HAASS C; WAGNER S L; TEPLOW D B; SISODIA S S (Reprint) ΑU
- CS JOHNS HOPKINS UNIV, SCH MED, NEUROPATHOL LAB, 558 ROSS RES BLDG, 720 RUTLAND AVE, BALTIMORE, MD, 21205 (Reprint); JOHNS HOPKINS UNIV, SCH MED. NEUROPATHOL LAB, BALTIMORE, MD, 21205; JOHNS HOPKINS UNIV, SCH MED, DEPT NEUROSCI, BALTIMORE, MD, 21205; JOHNS HOPKINS UNIV, SCH MED, DEPT PATHOL, BALTIMORE, MD, 21205; HARVARD UNIV, SCH MED, DEPT NEUROL, BOSTON, MA 02115; HARVARD UNIV, SCH MED, DEPT PATHOL, BOSTON, MA, 02115; BRIGHAM & WOMENS HOSP, CTR NEUROL DIS, BOSTON, MA, 02115; IND ASSOCIATES INC, SALK

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CYA
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     on STN
     94:671154 SCISEARCH
     The Genuine Article (R) Number: PM044
TT
     IDENTIFICATION OF THE MULTICATALYTIC ENZYME AS A POSSIBLE GAMMA-SECRETASE
     FOR THE AMYLOID PRECURSOR PROTEIN
AU
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CS
     BAYLOR COLL MED, DEPT NEUROL, HOUSTON, TX, 77030 (Reprint)
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GA
     The Genuine Article (R) Number: NU882
TI
     REVERSAL OF THE SWEDISH FAMILIAL
                                         ***ALZHEIMERS***
                                                              ***DISEASE***
     MUTANT PHENOTYPE IN CULTURED-CELLS TREATED WITH PHORBOL 12,13-DIBUTYRATE
ΑU
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      identifying compounds which inhibit A-beta peptide release and/or
      synthesis comprises an epitope tag within the A-beta sequence.
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PA
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